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Abstract: Objective: Previous research has yielded inconclusive results on the relationship between self-reported sleep quality and cognitive performance in healthy old age. Discrepant findings have been reported regarding processing speed and attention, executive functions, and episodic memory. However, sleep quality has also been found to be related to cognitive performance in patients with depression. Our aim was to clarify the relationship between sleep quality and cognitive performance in healthy older adults, and to evaluate the moderating role of subclinical depression on this relationship. Method: The Pittsburgh Sleep Quality Index was used to assess subjective sleep quality in 107 participants (age 61 years). A broad battery of neuropsychological tests measured basic cognitive processes, executive functions, and memory processes. Results: Subclinical depression moderated the link between sleep quality and cognitive performance. More precisely, poorer sleep quality was associated with lower performance in reasoning, semantic fluency, and shifting in those with high versus low levels of subclinical depression. Conclusion: Our findings suggest that poor sleep quality might affect higher order cognitive processes, particularly in those reporting higher levels of subclinical depression. Findings on the relationships between sleep quality, cognitive functioning, and depressive symptomatology are discussed in relation to neurobehavioral theories of sleep.

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Sleep quality and cognitive function in healthy old age: The moderating role of subclinical depression

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Abstract

Objective: Previous research has yielded inconclusive results on the relationship between self-reported sleep quality and cognitive performance in healthy old age. Discrepant findings have been reported regarding processing speed and attention, executive functions, and episodic memory. However, sleep quality has also been found to be related to cognitive performance in patients with depression. Our aim was to clarify the relationship between sleep quality and cognitive performance in healthy older adults, and to evaluate the moderating role of subclinical depression on this relationship.

Method: The Pittsburgh Sleep Quality Index (PSQI) was used to assess subjective sleep quality in 107 participants (age $\geq$ 61 years). A broad battery of neuropsychological tests measured basic cognitive processes, executive functions, and memory processes.

Results: Subclinical depression moderated the link between sleep quality and cognitive performance. More precisely, poorer sleep quality was associated with lower performance in reasoning, semantic fluency and shifting in those with high versus low levels of subclinical depression.

Conclusions: Our findings suggest that poor sleep quality might affect higher-order cognitive processes, particularly in those reporting higher levels of subclinical depression. Findings on the relationships between sleep quality, cognitive functioning and depressive symptomatology are discussed in relation to neuro-behavioral theories of sleep.

Key Words: Sleep quality – Aging – Cognition – Subclinical depression

Introduction

Sleep problems are among the most commonly reported health problems in the elderly, and the probability of experiencing sleep problems increases with advancing age. The average prevalence of sleep complaints in adulthood is between 8% and 18% (Asplund & Aberg, 1998); but in those 55 years and older, it is considerably higher (up to 41%), depending upon the number of medical conditions that are reported (Foley, Ancoli-Israel, Britz, & Walsh, 2004).

Sleep quality is thought to relate to cognitive functioning in old age. Indeed, studies have shown that poor sleep quality is associated with, for example, reduced executive functions (Nebes, Buysse, Halligan, Houck, & Monk, 2009). However, among the few studies that exist, results are inconclusive about the cognitive domains that are affected by self-reported sleep quality in healthy old age. Ambiguous findings have been reported regarding basic cognitive processes - such as processing speed, attention, and concentration - as well as regarding higher-order executive functions or episodic memory (Bastien et al., 2003; Nebes et al., 2009; Schmutte et al., 2007; Vignola, Lamoureux, Bastien, & Morin, 2000). Findings regarding which functions are affected are inconsistent. A positive relationship between self-reported sleep quality and cognitive functions has been reported in relation to processing speed (Bastien et al., 2003), executive functions (e.g., switching, divided attention, working memory, inhibition, verbal fluency, and problem solving, Nebes et al., 2009; Waters & Bucks, 2011) and long-term memory (Schmutte et al., 2007).

In accordance with these inconsistent findings, existing theoretical approaches focus on explaining sleep-related effects on specific cognitive domains. For example, the vigilance hypothesis proposes that objectively poor sleep is associated with a homeostatic drive for sleep, which can particularly lead to response slowing and lapses (Doran, Van Dongen, & Dinges, 2001). In fact, poor sleep has previously been found to impact performance on simple and complex reaction time tasks, attention and vigilance (Blatter et al., 2006; Buysse, Monk, Carrier, & Begley, 2005; for an overview see, Waters & Bucks, 2011). On the other hand, the frontal lobe hypothesis argues that sleep loss leads to transient modifications in the metabolism of the brain that particularly affects prefrontal cortex efficiency (Killgore, 2010). Accordingly, higher-order executive functions that involve prefrontal brain regions, to a great extent, are presumed to be strongly related to poor sleep quality (Braun et al., 1997; Durmer & Dinges, 2005; Jones & Harrison, 2001). This seems particularly true among older adults, given that prefrontal regions are highly age-sensitive; that is, age-related differences in cognitive performance are found primarily on tasks that depend upon the prefrontal cortex (MacPherson, Phillips, & Della Sala, 2002; West, 1996). So far, a general theory integrating the different cognitive domains is lacking. Moreover, existing theoretical approaches are primarily based upon objective sleep measures, whereas sleep-related effects on cognition in old age are based on subjectively-rated sleep quality.

It is important to note two things in the context of sleep and cognition in old age. First, subjective indicators of sleep quality are consistently related to measures of depression (Newman, Enright, Manolio, Haponik, & Wahl, 1997; Riemann, Berger, & Voderholzer, 2001). For example, Riemann et al. (2001) suggested that depression is related to sleep in a bi-directional way: sleep problems are symptoms of depression and, at the same time, can be a risk factor for depression. Second, Naismith et al. (2011) have shown that disturbed sleep in older patients with depression is related to cognitive functioning. That is, the duration of nocturnal awakenings has been found to be related to performance on verbal learning and memory, semantic fluency, response inhibition and problem-solving. Moreover, the authors suggest that the association between sleep and cognition in older patients with depression might be due to interference in neural circuits associated with mood, sleep and cognitive functions. They argue that particularly the fronto-subcortical circuitry (including the frontal cortex, striatum, globus pallidus and thalamus) could be essential, as it is important for the regulation of sleep-wake and affective states. For example, older adults with depression have been found to exhibit lesions in basal ganglia, as well as reduced volumes of the caudate nucleus, putamen and prefrontal cortex (cf. Naismith et al., 2003). Similarly, Levkovitz et al. (2009) identified improvement in the performance of previously-impaired cognitive domains (i.e., in sustained attention and cognitive planning) after transcranial magnetic stimulation over the prefrontal cortex in patients with depression, along with a significant reduction in depressive symptoms on the Hamilton Depression Rating Scale.

To take this potentially-moderating role of depression into account, previous studies on the link between sleep quality and cognitive performance in healthy older adults have used depression scores to exclude otherwise eligible participants (Bastien et al., 2003; Nebes et al., 2009; Vignola et al., 2000) and/or have controlled for the influence of (sub-)clinical depression (Nebes et al., 2009; Schmutte et al., 2007). In this study, we explicitly tested the potential moderating effect of subclinical depression on the relationship between sleep quality and cognitive function in old age. There are at least four reasons to anticipate this moderating effect. First, subsyndromal depression is highly prevalent in older adults, and this prevalence increases with advancing age (for two recent reviews, see Meeks, Vahia, Lavretsky, Kulkarni, & Jeste, 2011; VanItallie, 2005). Though not reaching a diagnostically-defined level, it is discussed as being of high clinical importance and associated with adverse health outcomes (Lyness, 2008) and cognitive impairments (Grabovich, Lu, Tang, Tu, & Lyness, 2010). Second, Waters and Bucks (2011) have conjectured that the relationship between sleep quality and cognitive performance might be stronger in some people than in others. One explanation for these individual differences in cognitive vulnerability could be depressive symptomatology. Third, studies focusing on the relationship between depression and cognition have specifically revealed impairments in working memory and set-shifting (Austin et al., 1999), two of the functions that have also been reported to be affected by sleep quality. Hence, including non-clinical depressive symptomatology as a moderator could lead to a stronger relationship between sleep quality and cognitive performance. Fourth, interactions between predictors (e.g., depressive symptoms × sleep quality) might be related to cognitive performance to a greater extent than either predictor alone. As suggested by Bassuk, Berkman, and Wypij (1998), individuals with depression may exhibit decreased cognitive performance due to motivational and attentiveness problems. An increase in the vulnerability of cognitive processes (Rabbitt, Donlan, Watson, McInnes, & Bent, 1995) to sleep quality might be the consequence (Vance, Roberson, McGuinness, & Fazeli, 2010).

In the present study, we aimed to clarify the relationship between self-reported sleep quality and cognitive performance in old age. We tested the hypothesis of a moderating role of subclinical depression on this relationship. We recruited our participants from a sample drawn from a healthy non-clinical population, excluding any who reported any severe medical condition (i.e., Parkinson's disease) and those with clinically-significant depression. Furthermore, we controlled for the influence of age and sleep medication, as previous studies have demonstrated that both variables are related to cognitive performance (Bastien et al., 2003; Paterniti, Dufouil, & Alperovitch, 2002; Waters & Bucks, 2011). Waters and Bucks (2011) have made the claim that interpreting previous sleep studies is difficult without controlling for age effects; and Paterniti et al. (2002) have reported that the long-term use of sleep medicines like benzodiazepines accentuates the decline in cognitive performance in old age. Moreover, using benzodiazepine leads to different relationships between sleep quality and cognitive performance (Bastien et al., 2003).

Method

Participants and Procedures

One-hundred-and-seven older adults (57% women; level of years of education: *M* = 10.0, *SD* = 2.3) ranging in age between 61 and 92 years (*M* = 72.0, *SD* = 5.7) participated in the study. Participants were recruited at a lecture for senior citizens at the University of

Zurich or through the distribution of flyers within seniors associations. The topic of the lecture was not related to sleep, depression, cognitive functions or any health issues. We excluded eleven otherwise eligible individuals due to incomplete information, indication of Parkinson's disease, a clinically-significant number of depressive symptoms based on their depression score (see test description below), or the usage of anti-depressive drugs, resulting in a final sample of 96 participants. Participants were in good health (i.e., as determined by self-reported subjective health) and none reported brain injuries, psycho-affective medication use, drug consumption or diseases affecting brain functioning. All participants were native German speakers. The study was approved by the local ethics committee and written informed consent was obtained from all participants. They received a personal performance profile and a voucher in the value of 10 CHF (approximately $10).

Informed consent, a demographic questionnaire and the sleep quality questionnaire were sent home to all participants. Individual testing took place at the research laboratory of the Department of Psychology at the University of Zurich and lasted, on average, about two and a half hours.

Measures

Sleep quality measure. Self-reported sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI, Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). This measure generally assesses sleep quality over the last month. However, in the German-language version (Riemann & Backhaus, 1996) that was used for this study, sleep quality is assessed with respect to the last *two weeks*. The PSQI consists of 19 questions (e.g., "During the past two weeks, how long (in minutes) has it usually taken you to fall asleep each night?") and seven subscales; i.e., subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of medication, and daytime dysfunction. The global PSQI score represents the sum of the seven subscales and ranges from 0 to 21. The PSQI does not ask about mood symptoms. The German-version of this sleep measure has been validated in healthy controls and insomnia patients, with a cut-off score of > 5, yielding a sensitivity of 98.7% and a specificity of 84.4% for primary insomnia. The internal consistency for the global score was $\alpha = .85$. In addition, the test-retest reliability over two weeks ($r = .87$) was high for the global score (Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002). For analyses, the total PSQI score was used. The PSQI scores in our sample are comparable to those of other studies (Buysse et al., 2008; Buysse et al., 1991).

Processing speed. We assessed processing speed with the Digit Symbol Substitution Test (Neuropsychological Aging Inventory, NAI, Oswald & Fleischmann, 2006). The Digit Symbol Substitution Test requires participants to fill as many empty boxes as possible by assigning nine simple symbols to each number (i.e., 1 to 9) within 90 seconds. The number of boxes filled correctly served as the outcome measure.

Verbal fluency. Semantic verbal fluency was assessed with a subtest of a German word fluency task (Regensburg Word Fluency Test, RWT, Aschenbrenner, Tucha, & Lange, 2000). Animal naming was assessed by asking participants to generate as many animals as possible. The number of correct non-redundant responses produced over three minutes was used in analyses.

Reasoning. Reasoning was assessed with subtest 3 of the German Achievement Measure System, using a version for adults over 50 years of age (Performance Test System, LPS 50+, Sturm, Willmes, & Horn, 1993). Participants had to identify the one irregularity in a sequence of eight abstract geometrical figures. They were asked to detect as many irregularities in each sequence as possible within a time limit of five minutes. The total number of sequences with the non-matching character correctly identified served as the outcome measure in subsequent analyses.

Set-Shifting. The Trail Making Test consists of two parts - form A and form B. In form A, participants are required to quickly combine 25 numbers in ascending order. In form B, participants are required to alternately combine numbers and letters in ascending order (e.g., 1, A, 2, B, 3, C, etc.). Subtracting the time to complete form A from that required to complete form B generated the final outcome measure (cf. Ble et al., 2005).

Inhibition. Computer testing of the Go/No-Go task was also implemented using the Tests of Attentional Performance (TAP 2.1, Zimmermann & Fimm, 2007) to measure inhibition. This test assesses one's ability to suppress a reaction triggered by external stimuli in favor of an internally-controlled behavior. The participants are required to respond as quickly as possible to an appropriate target (i.e., a lying cross, "x"), while controlling an inappropriate impulse (i.e., not responding to a fixed cross, "+"). Of the 40 stimuli that are presented, 20 are critical stimuli "x" and 20 are non-critical stimuli "+". For analyses, we used the numbers of correct responses and errors.

Episodic memory. Learning, free recall and recognition were assessed using the Verbal Learning and Memory Test (VLMT, Helmstaedter, Lendt, & Lux, 2001). The VLMT is the German version of the Rey Auditory Verbal Learning Test (RAVLT, Rey, 1958, 1964). A list of 15 words was read aloud by the experimenter five times. For the assessment of learning capacity, the participant was asked to repeat all the words on the list he or she could remember across all five trials. Further dimensions of long-term episodic memory - delayed free-recall and recognition - were collected after a 20-minute delay. Further variables that were used for analyses were the numbers of false positive responses (i.e., words that had not occurred in the list) and perseverations (i.e., repetitions of the same word, whether correct or not).

Depression questionnaire. We used the short-version of the German Geriatric Depression Scale (GDS, Sheikh & Yesavage, 1986) to assess depression. Possible scores range from 0 to 15. According to Gauggel and Birkner (1999) a score of ≥ 6 yields the best sensitivity and specificity for clinically-significant depression. Following this cut-off criterion, we excluded participants with a clinically-significant number of depressive symptoms from the study. Gauggel and Birkner reported an internal consistency of $\alpha = .91$.

This questionnaire does not ask about sleep problems. Scores on the GDS short version were slightly differently distributed in our sample than in other studies (e.g., Boey, 2000). However, this is not surprising, given that we excluded all participants with a GDS score of 6 or higher.

Statistical Analyses

As we used various cognitive measures, we wanted to reduce the number of variables. Therefore, we conducted an exploratory factor analysis of the 11 cognitive variables and applied promax rotation. Three factors with eigenvalues greater than one were extracted that explained together 59.97% of the total variance. The first factor contained the following tests: digit symbol substitution, animal naming, LPS reasoning, and Trail Making (form B minus form A), and was named *higher-order executive functions* ($\alpha = .75$). Three measures of the VLMT (i.e., learning capacity, free recall and recognition) loaded on the second factor; this factor was named *memory* ($\alpha = .85$). The remaining variables were loaded onto the third factor and named *inhibition* ($\alpha = .56$). This last factor included the number of correct responses and errors on the Go/No-Go test, as well as the number of false positive responses and perseverations on the VLMT.

Descriptive statistics and correlations among all variables of interest are shown in Table 1. To test whether sleep quality was related to performance on specific cognitive measures, three multiple regression models were generated for each factor (i.e., higher-order executive functions, memory, and inhibition). These regression models included age and usage of sleep medication as control variables, and sleep quality, subclinical depression, and the interaction between sleep quality and subclinical depression as predictors. The control variables and predictors were centered on their respective means and a term reflecting the interaction between sleep quality and depression was created (Aiken & West, 1991).

Insert Table 1 about here

Results

Higher-order executive functions factor. For this factor, the predictors explained 33% of the variance and the overall regression equation was significant; $F(5, 95) = 8.90$, $p < .001$. Sleep quality and subclinical depression were not significant; however, the interaction between sleep quality and subclinical depression reached significance, $t = 2.32$, $\beta = -.22$, $p < .05$. To disentangle the results of the cognitive processes associated with this factor, further analyses with the separate cognitive variables of the higher-order executive functions factor were conducted. The overall regression equation was significant for *LPS reasoning*, $F(5, 93) = 4.71$, $p < .001$. In this model, the predictors explained 21% of total variance. Sleep quality and subclinical depression were not significant predictors; however, a significant interaction was identified for the predictors sleep quality and subclinical depression, $t = 1.99$, $\beta = -.20$, $p < .05$. Simple slope tests then were conducted to clarify the nature of this interaction (see Figure 1). They revealed that, at higher levels of subclinical depression, poorer sleep quality was negatively related to performance on the LPS reasoning test, $t = 2.36$, $\beta = -.39$, $p < .05$. At lower levels of subclinical depression, poorer sleep quality was not related to LPS reasoning performance.

The same picture emerged for the *Animal Naming Test*: the overall regression equation was significant, $F(5, 95) = 4.93$, $p < .001$, and the predictors explained 21% of total variance. Again, no significant association was found for sleep quality and subclinical depression; however, a trend was found for the interaction between sleep quality and subclinical depression, $t = 1.74$, $\beta = -.18$, $p = .08$. Simple slope tests again revealed that, at higher levels of subclinical depression, poorer sleep quality was negatively related to performance on the Animal Naming Test, $t = 1.76$, $\beta = -.29$, $p = .08$ (see Figure 1). Conversely, at lower levels of subclinical depression, poorer sleep quality was not related to test performance.

The overall regression equation for the *Trail Making Test* was significance $F(5, 95) = 5.03$, $p < .001$. In this model, the predictors explained 22% of total variance. Sleep quality and subclinical depression were not significant, but a significant interaction between the predictor variables sleep quality and subclinical depression emerged, $t = 2.07$, $\beta = .21$, $p < .05$. Simple slope tests revealed that at higher levels of depression, poorer sleep quality was positively related to performance on the Trail Making Test, $t = 2.44$, $\beta = .39$, $p < .05$ (see Figure 1); whereas, once again, poorer sleep quality was not related to cognitive performance at lower levels of subclinical depression.

The overall regression equation for the *Digit Symbol Substitution Test* was significant $F(5, 95) = 4.08$, $p < .01$, and the predictors explained 18% of total variance. However, neither sleep quality, subclinical depression nor the interaction between these predictor variables achieved significance.

Overall, the findings of the higher-order executive functions factor indicate that only participants reporting higher levels of subclinical depression and poor sleep quality tended to exhibit lower levels of performance, whereas those with higher levels of subclinical depression but good sleep quality did not display any impaired performance on three of the four relevant tests.

Insert Figure 1 about here

Memory factor. The overall regression equation was significant $F(5, 95) = 4.39$, $p < .001$, $R^2 = .20$; however, neither sleep quality, subclinical depression nor the interaction between the predictors sleep quality and subclinical depression were significant components in the model.

Inhibition factor. The overall regression equation was non-significant ($p > .05$). Therefore, sleep-related variables were not interpreted.

Discussion

The present study was designed to clarify the relationship between self-reported sleep quality and cognitive performance in healthy older adults. A crucial component of our approach is that we considered the moderating role of subclinical depression on this relationship.

Our findings revealed that self-reported sleep quality is selectively related to cognitive performance in healthy old age. More precisely, a relationship to poorer sleep quality was identified for cognitive tasks assessing higher-order executive functions that involve an additional processing speed component. Importantly, when basic processing speed capabilities were measured alone, there was no significant association with sleep quality. Furthermore, no association was found with the inhibition or memory factors, each comprised of several cognitive measures.

Our finding that variables of higher-order executive functions factor are associated with poorer sleep quality is in line with the findings of Nebes et al. (2009). They found that good and poor sleepers differed in their performance of tasks measuring working memory, attentional set-shifting, and problem solving, but not tasks assessing processing speed or inhibition. This being said, in other studies an association with processing speed and inhibition was evident. For example, Bastien et al. (2003) found a relationship between processing speed and subjective sleep quality. However, this association involved performance on the Trail Making Test, parts A and B, which reflected higher-order executive functions in our study, while they also found no significant association with performance on the Digit Symbol Substitution Test. Regarding performance on inhibition tasks, Waters and Bucks (2011) reported an association between sleep deprivation and specific aspects of inhibition. More precisely, the authors stated that the intentional and controlled aspects of inhibition (e.g., measured by Negative Priming) are more affected than the automatic aspects (e.g., measured using the Stroop). Irrespective of this distinction, in our study, the inhibition factor included different measures and was not significantly related to sleep quality.

Also not affected by poor sleep quality was episodic memory performance, which also is consistent with previous research (Nebes et al., 2009; Vignola et al., 2000). However, in two different previous studies, an association between sleep quality and episodic memory performance was apparent (Bastien et al., 2003; Schmutte et al., 2007). Both of these studies used a multitude of subjective sleep measures (e.g., eight different variables were used in the study by Bastien et al., 2003) and only some of these variables correlated with memory performance. In contrast, in our study and in the study by Nebes et al. (2009), a composite score of sleep quality was used which could suggest that, using a more robust construct of sleep quality, there is no relationship to episodic memory performance in healthy old age.

To sum up, self-reported sleep quality in healthy older adults seems to be selectively related to higher-order executive functions. This finding lends support to previous studies that also identified a significant relationship between sleep quality and higher-order executive functions in elders (Bastien et al., 2003; Nebes et al., 2009; Schmutte et al., 2007) and it fits nicely with the frontal lobe hypothesis of sleep and cognition, stating that sleep loss particularly affects the frontal lobe and, accordingly, those functions associated with it (Harrison & Horne, 1998). Based upon these results, we suggest that a decrease in cognitive functioning related to poor sleep quality in healthy older adults might only occur when prefrontal brain areas are placed under exceptionally high demand; for example, through the simultaneous use of both higher- and lower-order cognitive processes. These high demands might be understood as some kind of cognitive complexity threshold that a task must reach before it can be affected by sleep quality. Accordingly, our findings might suggest that cognitive functions are not impaired by poor sleep quality, if the task only requires processing speed or simpler executive functions. However, as soon as cognitive demands increase, due to, for example, a time limit, poor sleep quality might be related to poorer cognitive performance in healthy older adults.

Besides the assumption that frontal brain regions are associated with the modulation of sleep processes and cognitive functioning, they also have been found to be involved in the regulation of mood (Monteleone & Maj, 2008; Waters & Bucks, 2011). In our study, the relationship between sleep quality and cognitive performance was limited to those participants reporting a higher level of subclinical depression, thereby exhibiting lower levels of cognitive performance. This is in agreement with what Naismith et al. (2011) reported - that disturbed sleep in older patients with clinically-relevant depression is related to cognitive functioning. The authors further suggested that fronto-subcortical circuitry could be essential to understanding the relationships between sleep quality, cognitive functioning, and depressive symptomatology. Applied to our cognitive complexity threshold hypothesis, our findings suggest that depressive symptomatology might lower this threshold. Consequently, poor sleep quality affects cognitive performance already in tasks of lower complexity, as long as they are dependent on the integrity of the prefrontal cortex. Accordingly, Naismith et al. (2011) who included patients with clinically-relevant depression, found that sleep quality was not only associated with highly-demanding executive functions, but also with processing speed and simpler inhibition tasks. Conversely, Benitez and Gunstad (2012) identified diminished cognitive functions with poor sleep quality, independent of depression. However, this inconsistent finding could be due to the fact that depression was measured with the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), while we measured it with the short form of the GDS. Furthermore, their participants included young adults, who might not express the same degree of depressive thoughts as older adults.

Combined with the results of our study, findings could indicate that the number of self-reported depressive symptoms modulates the cognitive complexity threshold through a fronto-subcortical circuit that regulates both mood and sleep-wake cycles. This threshold affects the impact that sleep quality has upon performance. Whereas in our study, older adults with subclinical depressive symptomatology revealed impairments only in higher-order cognitive functions including a time limit, in the study by Naismith et al. (2011), the presence of clinically-significant depression was associated with impaired performance on additional cognitive functions. According to Sedek, Brzezicka, and Von Hecker (2010), there is empirical evidence that a relationship between impaired cognition and subclinical depressive symptoms in older adults may be found only in tasks that permit the use of complex strategies. Our finding of selectively-impaired higher-order executive functions in association with subclinical depressive symptoms is in line with this observation. Moreover, we refine this relationship between cognitive impairment and depressive symptoms by considering the crucial role that sleep quality seems to play.

Our proposed cognitive complexity threshold theory fits well with Stern's Cognitive Reserve Hypothesis (Stern, 2002). According to Stern, cognitive reserve is defined as the amount of brain damage that can exist before cognitive impairment is evident. Accordingly, the model assumes the existence of a threshold, that once reached, inevitably leads to clinically-relevant cognitive impairment. This threshold is defined individually by the quantity of cognitive and brain reserve a person has, with greater reserves providing an elevated threshold before cognitive deficits emerge. Applied to our data, poor sleep quality *per se* seems not to lead to changes in cognitive performance. However, in interaction with higher levels of subclinical depression, our proposed cognitive complexity threshold is reduced, which leads to deficits in higher-order executive functions.

The present study has both limitations and implications for future research. One limitation might be that we measured each cognitive domain with just one or two single cognitive tests. Therefore, we cannot conclude that sleep quality is related to one specific cognitive domain. In order to address this question, future studies might want to focus on a more limited number of cognitive domains, but use a variety of tests measuring each cognitive domain. We attempted to reduce this limitation by performing factor analyses and statistically analyzing our data on the basis of latent cognitive factors. However, the four variables that formed the inhibition factor had relatively low reliability. Nevertheless, we decided to include these variables as one latent factor instead of four single variables to specifically examine the role of inhibitory functions and avoid multiple testing. Moreover, regarding episodic memory functions, it might be interesting to include some measure of long-term memory consolidation in future research, particularly because findings have revealed a positive impact of sleep on memory consolidation in young adults (e.g., Rasch, Buchel, Gais, & Born, 2007). Furthermore, another point to consider is that although we controlled for psychoactive medications, we did not assess the detailed usage of other medications. Therefore, the possibility remains that other medications or conditions (e.g., medication against pain, cardiac problems, or high blood pressure, etc.) might have affected sleep or mood. This should be addressed in future studies.

Future studies could focus on objective sleep measures assessed, for example, by performing actigraphy, electroencephalography (EEG) recorded during sleep, or diagnostic polysomnography. This would allow more detailed assessment of individual sleep quality and more precise definition of its components in poor and good sleepers and in (sub-clinically) depressed older adults. To the best of our knowledge, only two studies have used an objective sleep quality measure to examine cognitive differences in older, non-demented, community-dwelling individuals, all females (Blackwell et al., 2006; Yaffe, Blackwell, Barnes, Ancoli-Israel, & Stone, 2007). However, the cognitive tests were limited to the MMSE and Part B of the Trail-Making Test. Nonetheless, the findings are promising for future research as they revealed a significant relationship between these tests and objective sleep measures.

Objective measures and particularly diagnostic polysomnography also could include assessment for obstructive sleep apnea, as this condition is known to affect sleep quality. Obstructive sleep apnea refers to brief interruptions in breathing during sleep, which causes changes in blood oxygen saturation and sleep patterns. This, in turn, results in daytime sleepiness and changes in mood and cognition (Jackson, Howard, & Barnes, 2011). Sleepiness was not something we assessed in our study, but might be taken into consideration in future research.

To sum up, our findings reveal that particular higher-order executive functions involving speed seem to be related to self-reported sleep quality in healthy older adults. However, it also seems important to consider low levels of depressive symptomatology together with sleep quality, as they appear to be interrelated.

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Table 1. Descriptive Statistics and Correlations Among the Measured Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Digit Symbol Substitution	-														
2. Animal naming	.419***	-													
3. LPS reasoning	.527***	.399***	-												
4. Trail Making (B – A)	-.454***	-.437***	-.418***	-											
5. Go/No-Go correct	.034	-.024	.142	-.148	-										
6. Go/No-Go errors	-.071	-.044	-.181	.146	-.552***	-									
7. Learning	.387***	.468***	.329***	-.332***	-.045	-.105	-								
8. Free recall	.366***	.386***	.335***	-.337***	-.059	-.019	.819***	-							
9. Recognition	.065	.297**	.109	-.151	-.058	.061	.549***	.598***	-						
10. False positive	-.039	-.091	-.177	.263*	-.306**	.113	-.151	-.140	-.095	-					
11. Perseveration	.145	.048	-.108	-.016	-.126	.193	.140	.104	.132	086	-				
12. Age	-.373***	-.361***	-.394***	.362***	-.069	.070	-.407***	.385***	.280**	.124	-.088	-			
13. Sleep medication	-.142	-.245*	-.112	.154	-.079	-.068	-.091	-.120	-.255*	.147	-.032	.094	-		
14. Depression	-.153	-.117	-.030	.137	-.023	-.067	-.049	-.098	-.081	.055	-.142	-.049	.255*	-	
15. Sleep quality	-.120	-.281**	-.210*	.280**	-.039	-.073	-.159	-.234*	-.227*	.078	-.186	.204	609***	.405***	-
M	43.09	41.45	21.53	57.51	19.78	1.70	48.49	10.03	13.65	1.22	2.89	71.73	.31	1.13	5.17
SD	10.72	10.35	4.84	28.31	.58	2.25	10.55	3.46	1.51	1.98	3.17	5.66	.812	1.16	3.40
Range	17 - 67	13 - 63	12 - 35	13 - 121	17 - 20	0-13	20 - 71	2 - 15	8 - 15	0 - 13	0 - 14	61 - 92	0 - 3	0 - 4	0 - 16

Note. $N = 96$; *$p < .05$, **$p < .01$, ***$p < .001$.

Figure Captions

Figure 1. LPS reasoning (accuracy), Animal Naming Test (accuracy) and Trail Making Test (reaction time; B –A) as a function of sleep quality and subclinical depression.